Conferences and Reviews

Dizzy Patients
Diagnosis and Treatment

Discussant
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This discussion was selected from the weekly Grand Rounds in the Department of Medicine, University of Washington School of Medicine, Seattle. Taken from a transcription, it has been edited by Jonathan Drachman, MD, Chief Medical Resident; Henry Rosen, MD, Professor and Associate Chair; and Paul Ramsey, MD, Professor and Chair of the Department of Medicine.

Two years after his historic orbital space flight, and in the middle of his campaign for the United States Senate, Lieutenant Colonel John H. Glenn Jr accidentally fell and struck the left side of his head on a bathtub, causing disabling dizziness and nausea that confined him to bed for months and eventually forced him to pull out of the Senate race (R. Witkin, "Glenn Quits Race in Ohio, Citing His Slow Recovery," *New York Times*, March 31, 1964, pp 1, 18, 19). At a news conference announcing his withdrawal, a panel of physicians reassured the world that Glenn's dizziness, despite rumors, was unrelated to space travel but instead resulted from trauma to the inner ear, a relatively routine condition that only time would heal. As if to challenge the notion that dizziness was untreatable, hundreds of dizzy patients then wrote to the colonel, expressing sympathy and encouraging him to try a variety of cures, each provided with its own testimonial: "Drink whiskey before going to work," "Take vitamin B_{12} or vitamin C," "Drink large volumes of pineapple juice," "Ask for electric shocks behind the ear," "Read the book *I Believe in Miracles*," "Attach a gyroscope to the hip," and "Whatever else (the colonel does), stay away from those little blue and white pills Antivert [meclizine hydrochloride]."[1]

These folk remedies are a reminder that dizziness, despite its prevalence, often frustrates and confounds both physicians and patients. Dizziness accounts for 1% of visits to US office-based physicians.[2] Of patients older than 60 years, 20% have experienced dizziness severe enough to affect their daily activities.[3,4] Among dizzy patients who seek help, almost 70% initially see general internists or family practitioners, only 4% are referred to specialists, and 90% leave the physician's office with a prescription, most often for meclizine.[2] In this review I discuss the common causes of dizziness and provide a simple yet scientific approach to diagnosis and management.*

*See also the editorial by K. Kroenke, MD, "Dizziness in Primary Care," on pages 73-74 of this issue.

Diagnosis

Table 1, which is compiled from four prospective studies on dizziness,[5-8] emphasizes several points. First, among patients with dizziness who consult general practitioners, three diagnostic groups account for more than 75% of the final diagnoses: peripheral vestibular disorders, psychiatric disorders, and multiple sensory deficits.[5,6] Second, central vestibular disorders—such as brain-stem and cerebellar ischemia—may cause as much as 23% of cases of dizziness in older patients.[7] Third, hypotension is an uncommon cause of chronic dizziness in the clinic setting, but is responsible for 16% of the cases of dizziness seen in emergency departments.[8] Finally, only 8% to 14% of cases remain undiagnosed after thorough evaluation.

Peripheral Vestibular Disorders

Vertigo is a type of dizziness characterized by sensations of movement (twirling, tumbling, or tilting) felt inside a person's head. Vestibular disorders, either peripheral (inner ear) or central (brain stem, cerebellum), all cause vertigo that is episodic and of abrupt onset. Helpful distinguishing features are the duration of vertigo, the precipitating factors, and the associated symptoms.

Patients with benign positional vertigo (BPV) describe episodic vertigo lasting less than a minute, brought on by movements of the head—such as rolling over in bed, hyperextension of the neck, or bending over—and without other associated symptoms.[9,10] In all, 15% to 50% have had recent ear trauma or infection.[9-13]

The Hallpike-Dix maneuver, historically the key diagnostic test of BPV, is illustrated and described in Figure 1.[10-16] Although a positive test is usually required for diagnosis, the sensitivity of the maneuver was only 60% in one study of 250 patients with symptoms of BPV.[17] Of the initially negative tests, 30% became positive during subsequent clinic visits,[17] and once documented, a positive test was not present during every visit.[9] Few elderly pa-

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TABLE 1.—*Causes of Dizziness in 4 Studies*

Cause, %*	Study: Drachman and Hart[5] (n = 102)	Kroenke et al[6] (n = 100)	Sloane and Baloh[7] (n = 116)	Herr, et al[8] (n = 125)
Setting	Dizziness clinic	Walk-in clinic	Neurotology clinic†	Emergency department
Peripheral vestibular disease	38	43	46	43
Benign positional vertigo	12	16	23	--
Vestibular neuronitis	9	3	8	--
Meniere's syndrome	3	4	8	--
Other	14	20	7	--
Psychiatric	24	16	9	11
Multiple sensory deficit	13	2 (17)‡	2	1
Central vestibular disorder	7	10	23	6
Cardiovascular disease§	4	6	3	16
Unknown	9	8	14	10

*Totals do not equal 100% because miscellaneous diagnoses are excluded.
†Aged 70 and older.
‡Multiple sensory deficits were primary cause in 2% and primary or contributory cause in 17%.
§Hypotension—for example, orthostatic, vasovagal, arrhythmia-induced.

tients tolerate hyperextension of the head over the table edge; therefore, many clinicians allow a patient's head to land squarely on the table. Movement from seated to supine should occur over two seconds, with slower movements reducing the sensitivity of the test.[18]

Aside from confirming the diagnosis of BPV, a positive Hallpike-Dix maneuver also distinguishes BPV from central positional vertigo, an uncommon disorder usually caused by cerebellar tumors, carcinomatous cerebellar degeneration, or multiple sclerosis.[19-21] Table 2 reviews the three features—latency, adaptation, and fatigability—that may be helpful in differentiating these disorders.

Benign positional vertigo is commonly postulated to result from damage to the delicate sensory units of the inner ear, the semicircular canals and otolith organs. The three semicircular canals (anterior, posterior, and horizontal) normally detect spinning movements (that is, angular acceleration). Hair cells in each canal respond to rotation in a plane parallel to the canal by bending with the endolymph currents generated. The sensory units of the two otolith organs, saccule and utricle, differ in that they are denser than the surrounding endolymph. Embedded in a gelatinous membrane coated with calcium carbonate crystals (otoliths), these hair cells bend with gravitational forces (that is, linear acceleration), enabling the detection of positions or movements that tilt away from the vertical or horizontal planes. In patients with BPV, otoliths from the utricle become dislodged, perhaps because of recent ear trauma or degenerative changes, and settle to the most dependent portion of the inner ear, the posterior semicircular canal. With the posterior canal now abnormally burdened by otoliths, any movement in a plane parallel to the posterior canal (such as with the Hallpike-Dix maneuver) will produce exaggerated movement of the hair cells in that canal, provoking vertigo and nystagmus. The several-second latent period that is observed (Table 2) may reflect the time necessary to generate these exaggerated endolymph currents. "Fatigability" may occur because repeated head movements disperse otoliths throughout the endolymph system and away from the posterior canal.[18]

Evidence that BPV arises from the posterior semicircular canal includes the following: stimulation of the undermost posterior canal produces a nystagmus identical to that observed in BPV (Table 3)[22(p51)]; in patients with BPV, otoliths have been found in the posterior semicircular canal[23,24]; and patients with disabling BPV are cured after plugging or neurectomy of the posterior canal.[9,25]

Patients with vestibular neuronitis experience abrupt sustained vertigo lasting one to seven days, associated in 25% to 50% with a recent febrile illness that is usually diagnosed as a respiratory tract virus.[12,26] Examination reveals decreased caloric responses in the affected ear and a spontaneous rotatory nystagmus.[12,26,27] The term "vestibular neuronitis" is preferred to the more traditional "labyrinthitis" because hearing loss and tinnitus are absent (the labyrinth includes both the vestibular and cochlear organs).

In patients who unexpectedly died soon after an episode of vestibular neuronitis, pathologic studies of the affected ear demonstrated atrophy of the vestibular nerve with normal sensory organs and blood supply.[28] The observation of identical findings in a patient with herpes zoster oticus and vertigo, together with the frequently observed fever in vestibular neuronitis, has led to the suspi-

TABLE 2.—*Distinguishing Benign From Central Positional Vertigo*

Feature	Benign Positional Vertigo	Central Positional Vertigo
Latent period*	Yes, 2-20 seconds	No
Adaptation†	Yes, lasts <50 seconds	No, nystagmus persists
Fatigability‡	Yes, disappears on repetition	No, persists

*Is there a pause between assuming the supine position and the appearance of vertigo or nystagmus?
†Does the central nervous system *adapt* and extinguish the nystagmus?
‡Does prompt repetition of the maneuver result in weaker and briefer nystagmus?

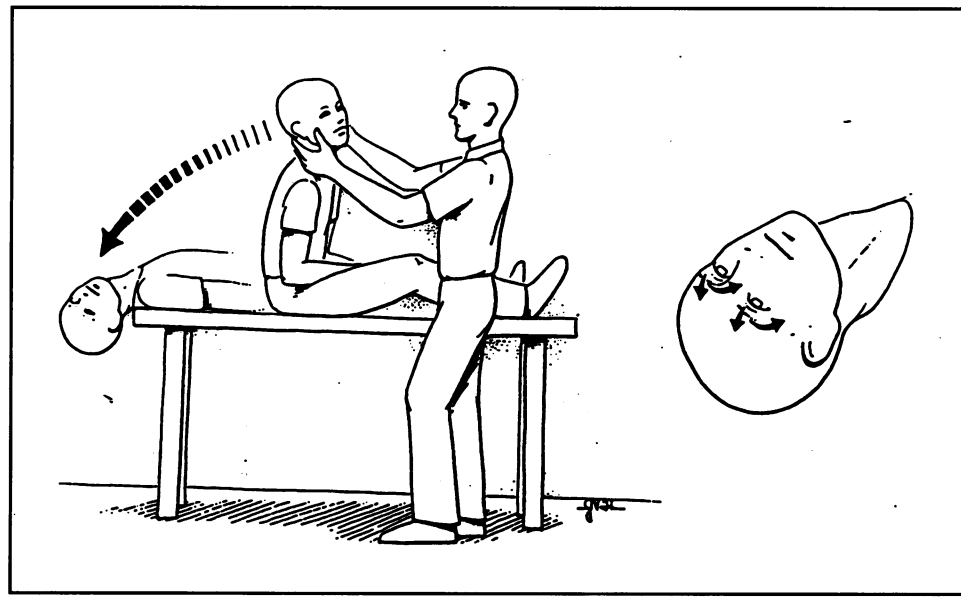

Figure 1.—The Hallpike-Dix maneuver: With the patient seated, the examiner turns the patient's head to one side and directs the patient to lie down, hyperextending the head over the edge of the table. In a positive test, a temporary vertigo and rotatory nystagmus will occur after several seconds, with the quick component of the superior pole of the eye rotating toward the undermost ear. If the test is negative, the examiner should repeat the test with the head turned to the opposite side (adapted from Mohr[10]). Although often referred to as the "Nylen-Barany" test, the designation "Hallpike-Dix" test is more appropriate. Barany and Nylen described the occurrence of benign positional vertigo in the 1920s, but emphasized that position, not movement, of the head was most important.[11,14-16] In a 1952 paper summarizing the findings of 100 patients with benign positional vertigo, Hallpike and Dix emphasized the importance of movement and specifically described the maneuver used today.[12]

cion, never proved, that vestibular neuronitis results from a viral infection.

Meniere's syndrome is a disorder named after Prosper Menière who was the first to attribute vertigo to diseases of the inner ear.[29] The syndrome is associated with vertigo that lasts hours, and, in contrast to other peripheral vestibular disorders, affected patients have cochlear symptoms—neurosensory hearing loss, tinnitus, and ear fullness. Sounds are distorted and tinny to the affected ear, and given that the opposite ear is usually normal, pure tones may be perceived as two separate notes (diplacusis). The characteristic disorder was identified in 1938 in two patients who died during surgical therapy for Meniere's syndrome: the affected ears revealed a massively distended endolymphatic system (endolymphatic hydrops) with herniated and ruptured endolymphatic membranes.[30]

The rotatory nystagmus that is characteristic of unilateral peripheral vestibular disorders (for example, vestibular neuronitis, Meniere's syndrome) reflects the specific connections between the semicircular canals and eye muscles. During head movements, the normal vestibular system stabilizes the retinal image by moving the eyes exactly opposite the head movement. Without this vestibulo-ocular reflex, it would be impossible to focus on objects when walking, riding, or even breathing. The accuracy and efficiency of this reflex can be easily demonstrated by holding a pencil vertically in front of the face and moving it from side to side through a 10-degree arc, five times per second. The pencil will appear blurred because the retina cannot compensate quickly enough for the shifting image. If the experiment is repeated with the pencil stationary and the head moved back and forth through the same arc and with the same frequency, the pencil will remain sharply defined. The eye movements are identical in the two examples, but only in the second experiment are the semicircular canals being used to keep the pencil in focus.[31]

To maintain this stable retinal image, each semicircular canal is linked to two eye muscles, one on each side, that move both eyes in a plane parallel to the canal and opposite the head movement (Table 3). In patients with unilateral peripheral vestibular disorders, an imbalance between the right and left sides allows three ipsilateral and three contralateral muscles to act as if unopposed. Because the normal actions of most of these muscles (all except the medial and lateral recti) will rotate the eye medially or laterally, the eye movements produced in uni-

TABLE 3.—*Connections Between Semicircular Canals and Eye Muscles*

Semicircular Canal	*Ipsilateral*	*Contralateral*
Anterior	Superior rectus	Inferior oblique
Horizontal	Medial rectus	Lateral rectus
Posterior	Superior oblique	Inferior rectus

*From Baloh and Honrubia.[22]

lateral vestibular disease are complicated and always include rotatory components. Pure horizontal or vertical nystagmus is not characteristic of peripheral vestibular disease and should suggest a central cause.

Central Vestibular Disorders

In a classic paper that tabulated the symptoms of 500 different patients with strokes, dizziness was a rare symptom in anterior circulation disease (internal carotid, middle cerebral, anterior cerebral arteries), but affected 75% to 100% with posterior circulation disease (basilar, anterior and posterior inferior cerebellar, vertebral arteries).[32] In 75% of those with dizziness and posterior circulation disease, the diagnosis of stroke was obvious because the dizziness either accompanied or appeared after other neurologic symptoms. In patients with brain-stem ischemia, the symptoms accompanying dizziness were usually dysarthria, numbness, diplopia, or hemiparesis.[32] Cerebellar ischemia, however, may be more difficult to distinguish from peripheral vestibular disease (such as vestibular neuronitis); the associated neurologic finding that suggests a cerebellar cause may be as subtle as a mild dysarthria, vertical nystagmus, dysmetria in one arm instead of two, or early findings of a posterior fossal mass, such as headache or depressed mental status.[33-35] If the direction of the nystagmus is opposite the direction that the patient falls, some think cerebellar ischemia is more likely than peripheral vestibular disease,[33] although exceptions occur.[34]

In 25% of patients with dizziness and posterior circulation disease, dizziness is the sole initial symptom, although an important note is that the other neurologic clues listed earlier usually appear within six weeks.[32] It was concluded that isolated dizziness persisting longer than six weeks rarely results from cerebrovascular disease. In support of this conclusion, another study of 86 patients with idiopathic episodic vertigo (lasting 5 minutes to 24 hours) found no cases of vertebrobasilar ischemia at 3.5 years' follow-up (83% were better, 10% had Meniere's disease, and 7% had BPV).[36]

Multiple Sensory Deficits

Combinations of visual impairment, neuropathy, and vestibular deficits may deprive patients of just enough sensory information that they begin to experience dizziness. These patients, usually elderly, have no dizziness at rest but feel vague unsteadiness when walking, especially in unfamiliar environments or when executing turns. A useful diagnostic test in the office is to have the patient make several right-angle turns when walking. A patient with multiple sensory deficits may begin to feel dizzy, a sensation immediately relieved by additional proprioceptive information, such as allowing the patient to touch the wall or table.

The Mercury and Gemini astronauts experienced no dizziness during flight, presumably because they were strapped to their seats for the entire voyage. During the Apollo, Spacelab, and Shuttle programs, however, astronauts frequently moved around in the weightless environment, depriving themselves of familiar proprioceptive information and sometimes inducing feelings of disequilibrium. This dizziness would resolve, just as in patients with multiple sensory deficits, after simply touching the cabin wall.[37,38]

Clinical Approach

A careful patient interview elicits the diagnosis in at least 75% of patients.[6,7] Open-ended questions help classify the dizziness as *vertigo*, the sensation of movement in the head, implying peripheral or central vestibular disease; *impending faint* (presyncope), implying hypotension; or *disequilibrium*, unsteadiness not in the head, implying cerebellar or proprioceptive disorders. The complaints of some patients, however (40% in one study), may be too vague or diverse to confine to a single category, despite a clinician's best efforts.[7] In patients with vertigo, helpful distinguishing features are the duration of vertigo (BPV, seconds; Meniere's, hours; vestibular neuronitis, days; vertebrobasilar ischemia, minutes to days), precipitating factors (positional or spontaneous), and associated symptoms (cochlear symptoms, fever, head trauma, other neurologic symptoms). The cause of dizziness may become obvious after a careful review of the patient's exposures (welder's fumes, carbon monoxide, hydrocarbon solvents) and medications (quinine, alcohol, antihypertensives, anticonvulsants, antidepressants, cardiovascular medications).[39-41]

Diseases that affect both inner ears equally, such as streptomycin toxicity, do not cause vertigo, because vertigo requires an imbalance between the right and left sides. Instead, patients with bilateral vestibular disease experience a sensation called "oscillopsia" (inability to focus vision during head movements). When patients lack a functional vestibulo-ocular reflex, their environment appears to bounce around just as a video picture jumps around when the camera is carried on a photographer's shoulder.[42]

The physical examination should focus on a patient's vital signs, ears, heart, and nervous system. Nystagmus that appears only on extreme lateral gaze is not pathologic, as it occurs in 60% of normal people.[43] Nystagmus that occurs when the eyes are closer to the primary eye position is abnormal: rotatory nystagmus suggests peripheral or central vestibular disease, and pure horizontal or vertical nystagmus implies a central cause. A positive Romberg test (pronounced increase in body swaying with eyes closed compared with eyes open) suggests proprioceptive disease, not cerebellar disease as is commonly thought.[44] The Hallpike-Dix maneuver (Figure 1) is indicated in patients with positional vertigo. If the clinician suspects multiple sensory deficits, the patient should be observed while walking and making sudden turns. Although many clinicians think that dizziness aggravated by the hyperventilation maneuver identifies a specific "hyperventilation syndrome,"[5] this maneuver may be less specific than previously thought, with some studies showing that as much as 20% of cases of dizziness from all causes (including peripheral vestibular disorders) worsen during forced hyperventilation.[6,8]

Measuring the blood count and electrolyte levels is appropriate in patients with orthostatic hypotension and as a screen for gastrointestinal bleeding, prerenal azotemia, and adrenal insufficiency. In patients with vertigo accompanied by cochlear symptoms, prompt referral for audiometry will address the possibility of Meniere's syndrome. Other laboratory tests are rarely helpful.[6] Electronystagmograms, though commonly done in research studies, rarely contribute new clinical information. Although 65% of electronystagmograms were abnormal in one study of 75 dizzy patients, the positive findings usually confirmed diagnoses obvious from the history and physical examination alone.[45] Furthermore, the clinical observation of nystagmus is just as accurate as electronystagmography in patients with BPV.[17]

Management

Both anticholinergic (scopolamine) and antihistamine (dimenhydrinate, meclizine) medications are commonly used to treat vertigo (astronauts combine scopolamine with amphetamines), although there are few drug trials that have a double-blind placebo design. In motion-sickness experiments in which more than 16,000 soldiers were enrolled, dimenhydrinate and meclizine were more effective than placebo.[46] In human volunteers subjected to rotational accelerations, both scopolamine and dimenhydrinate prevented postrotational vertigo, nausea, and nystagmus better than placebo.[47,48] There are only two positive double-blind placebo trials in dizzy patients, both of short duration: meclizine and dimenhydrinate were each effective in 50 elderly patients with dizziness of diverse causes,[49] and meclizine was effective in 31 patients with vertigo from various causes.[50] Benzodiazepines were ineffective in the treatment of BPV.[51]

Scopolamine and dimenhydrinate have traditionally been used for motion sickness and meclizine for dizziness, though there is no reason why these medications should not be interchangeable. The peak efficacy of the scopolamine patch does not appear until eight hours after application, making it most useful as a prophylactic medication.[52]

Active exercise will expedite the recovery from vertigo. The central nervous system has a remarkable ability to adapt and extinguish any disequilibrium that results from sensory conflicts between the right and left vestibular systems, between the semicircular canals and otolith organs, or between the vestibular and proprioceptive systems.[53] Such adaptation explains why motion sickness resolves on long voyages, why space sickness diminishes after 48 hours of flight, and why dancers can learn to suppress caloric-induced nystagmus.[54] Adaptation occurs more quickly with active exercise. Squirrel monkeys (*Saimiri sciureus*) subjected to unilateral labyrinthectomy are much less ataxic if exercised daily in rotating cages.[55] Humans who actively exercise also recover much more quickly from the vertigo and nystagmus that develop after unilateral labyrinthectomy.[54,56-58] In one study of 67 patients with BPV, 66 were cured after a week of performing simple exercises several times a day.[59]

The management of patients with Meniere's syndrome has focused on attempts to reduce the distention of the endolymphatic space. One double-blind randomized trial of 26 patients with Meniere's syndrome compared a regimen of hydrochlorothiazide, 25 mg three times a day, with placebo.[60] At one year follow-up, the use of hydrochlorothiazide resulted in a substantial reduction of vertigo and hearing loss (there was no difference in the incidence of tinnitus between the two groups). Betahistine hydrochloride, a histamine H_1-agonist that is generally unavailable, was superior to placebo in two studies.[61,62]

Endolymphatic shunt surgery, despite its theoretical attractiveness, was no more effective than a sham procedure (mastoidectomy); 75% of both treatment groups were improved at three years' follow-up.[63,64] In another study of 123 patients with unilateral Meniere's syndrome, the outcome in patients after an endolymphatic shunt operation was no better than in those who refused an operation—60% of both groups were improved at two years' follow-up.[65] Despite these results, which some dispute,[66] the endolymphatic shunt operation is still preferred by a third of US physicians as the initial operation for Meniere's syndrome.[67] Other surgical procedures, such as vestibular neurectomy and labyrinthectomy, are also carried out for Meniere's syndrome, although none have been subjected to a randomized trial.[68]

Patients with multiple sensory deficits may benefit from measures that provide them more sensory information about their environment, such as cataract extraction, appropriate eyeglass prescription, a night light at home, or physical therapy for learning to use a cane or walker.

Prognosis

The prognosis in patients with dizziness is good, with rates of hospital admission, institutionalization, and death no different from those of age-matched controls.[3,4] With follow-up periods varying from 6 months to 12 years, the dizziness improves or resolves in 75% of patients with Meniere's syndrome,[63-65] 83% of patients with idiopathic vertigo,[36] 98% of patients with BPV,[59] and 90% to 100% of patients with vestibular neuronitis.[26,27]

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